AD 297 348

AMRL-TDR-62-130

297 348

STUDIES AT MODERATE HYPOTHERMIC TEMPERATURES OF FACTORS AFFECTING SURVIVAL UNDER PROLONGED HYPOTHERMIA

TECHNICAL DOCUMENTARY REPORT No. AMRL-TDR-62-130

NOVEMBER 1962

BIOMEDICAL LABORATORY
6570th AEROSPACE MEDICAL RESEARCH LABORATORIES
AEROSPACE MEDICAL DIVISION
AIR FORCE SYSTEM COMMAND
WRIGHT-PATTERSON AIR FORCE BASE, OHIO

Contract Monitor: J. F. Hall, Jr.
Project No. 7222, Task No. 722204

(Prepared under Contract No. AF 33(616)-6767
by
Evangelos T. Angelakos, M.D., Ph.D.
Boston University School of Medicine)

NOTICES

When US Government drawings, specifications, or other data are used for any purpose other than a definitely related government procurement operation, the government thereby incurs no responsibility nor any obligation whatsoever; and the fact that the government may have formulated, furnished, or in any way supplied the said drawings, specifications, or other data is not to be regarded by implication or otherwise, as in any manner licensing the holder or any other person or corporation, or conveying any rights or permission to manufacture, use, or sell any patented invention that may in any way be related thereto.

Qualified requesters may obtain copies from ASTIA. Orders will be expedited if placed through the librarian or other person designated to request documents from ASTIA.

Do not return this copy. Retain or destroy.

Stock quantities available at Office of Technical Services, Department of Commerce, $.75.

800 - February 1963 - 4-27

FOREWORD

This study was initiated by the Biomedical Laboratory of 6570th Aerospace Medical Research Laboratories, Aerospace Medical Division, Wright-Patterson Air Force Base, Ohio. The research was conducted in the Hypothermia Laboratory at the Department of Physiology of Boston University School of Medicine under Contract No. AF 33(616)-6767. Dr. E.T. Angelakos was the principal investigator for Boston University School of Medicine. The work was performed in support of Project No. 7222 "Biophysics in Flight" and Task No. 722204 "Human Thermal Stress in Extended Environment." The research sponsored by this contract started in July 1, 1960 and was completed on June 30, 1962.

The author wishes to acknowledge the collaboration of the following individuals who contributed to the project. Dr. A.H. Hegnauer, Professor of Physiology, acted as consultant. Dr. J.C. Torres, Instructor in Physiology, assisted in certain phases of the program. Mr. Donald Rippon, Technician, assisted in the performance of the experiments.

The experiments reported herein were conducted according to the "Principles of Laboratory Animal Care" established by the National Society for Medical Research.

ABSTRACT

The mortality of anesthetized dogs maintained under hypothermia for many hours was studied, and the factors responsible for death under these conditions were evaluated. Data are presented on technically successful experiments in 40 dogs maintained at 26 ± 1°C. Of these, 25% survived a period of 18 to 20 hours and were subsequently rewarmed while 17.5% rewarmed spontaneously. Of the remaining, 45% died in prolonged hypothermia, 10% in ventricular fibrillation, and 35% in cardiac standstill. In addition, a total of 12.5% of the animals died during rewarming. Among the animals that died during maintained hypothermia, mortality was progressive and there seemed to be no critical time. The median lethal time at 26 ± 1°C is estimated to be 18 hours.

There was a progressive decrease in the average normal pacemaker heart rate in animals maintained at a relatively fixed hypothermic temperature. Values of arterial hematocrit showed a progressive and striking increase during cooling, which was maintained under hypothermia, and during rewarming. The average values from 14 animals before cooling, at the start and end of prolonged hypothermia, and after rewarming were: 44, 52, 61, and 64, respectively.

The degree of hypothermic acidosis was not altered consistently during maintained hypothermia. Plasma electrolyte changes were inconsistent and could not be interpreted without data on plasma volume.

It is concluded that the factors that limit survival under prolonged hypothermia are different from those involved in the acute induction of the hypothermic state. Changes in the cardiac pacemaker rate may play a significant role.

The data indicate the need for additional studies in order to further elucidate the factors affecting survival under maintained hypothermia and to establish their relative significance.

PUBLICATION REVIEW

This technical documentary report has been reviewed and is approved.

Jos. M. Quashnock
Colonel, USAF, MC
Chief, Biomedical Laboratory

TABLE OF CONTENTS

LIST OF TABLES

STUDIES AT MODERATE HYPOTHERMIC TEMPERATURES OF FACTORS AFFECTING SURVIVAL UNDER PROLONGED HYPOTHERMIA

INTRODUCTION

The purpose of this investigation was to study the mortality of anesthetized dogs maintained under hypothermia for many hours, and to evaluate the factors responsible for death under these conditions.

Most of the available studies on hypothermic mortality (1,2,3) deal primarily with the acute effects of a progressive lowering of body temperature. Relatively little is known about the effects of low temperatures when they are maintained for long periods of time. Yet, such information is of great fundamental and practical value.

The effects of acute hypothermia reflect in large part the reactions of the organism to a drastic change and therefore cannot be taken as a reliable indication of the effects of cold under steady state conditions. The latter can be studied only under conditions of maintained hypothermia at a constant temperature. From a practical point of view, a thorough understanding of the effects of prolonged hypothermia may lead to such applications as the suggested use of hypothermia for space travel and protection from lethal radiations.

Experiments on prolonged hypothermia are difficult to perform due to multitude of technical problems that arise in connection with the maintenance of anesthetized animals, and the collection of experimental data, for periods longer than 8 hours.

When studies on prolonged hypothermia were first initiated in this laboratory, an attempt was made to maintain animals which were cooled to deep hypothermic temperatures (less than 18°C) for a period of 24 hours or more. However, it soon became apparent that information was first needed on the effects of prolonged hypothermia at moderate hypothermic temperatures (near 25°C). Therefore a series of experiments was designed in which observations were made in animals maintained at 26 $\pm$ 1°C for a period of 18-20 hours. Results of these studies performed over the past 2 years are summarized in this report.

METHODS

All studies were made on mongrel dogs initially anesthetized with pentobarbital (33 mg/kg).* In most cases the animals were cooled by being enclosed in a "hypothermic blanket" (Therm-O-Rite Products, Inc.) through which a cooling fluid was circulated. Cooling was induced first rapidly to a body temperature of 28°C. The animals were then stabilized at a temperature of 26 ± 1°C. In the successful experiments, this temperature was maintained for a period of 18-20 hours. Subsequently, the surviving animals were rewarmed and allowed to recover. Several of the survivors were also kept for a week or more for observation.

Body temperature was measured from a thermistor probe inserted in the esophagous at the level of the heart. Generally, blood pressure was measured electromanometrically from the catheterized left carotid artery and recorded on one channel of a Sanborn Twin-Viso Recorder. The electrocardiogram (lead II) was recorded on the other channel. Continuous records were taken throughout the experimental period of cooling, maintaining under hypothermia, and rewarming. The entire period was approximately 30 hours.

Arterial blood electrolytes and pH were measured using conventional techniques. Measurements of pH were made at the existing body temperatures using a unit made by Instrumentation Laboratories, Inc. Blood electrolytes were determined on samples of serum. Sodium and potassium were determined with the flame photometer, chloride with the Cotlove chloridometer (4) and calcium with the microanalytic method of Bachra et al (5).

RESULTS

A. Survival Under Prolonged Hypothermia

Data on survival under prolonged hypothermia were obtained from 40 animals and are summarized in Table 1. Animals that survived for periods of 18 to 20 hours at 26 ± 1°C were subsequently rewarmed. A total of 15 out of 40 (37.5%) survived the period of prolonged hypothermia, but 5 out of these (12.5%) died during rewarming.

The majority of the animals were maintained at the desired hypothermic temperature without the necessity of any further cooling. However 7 out of 40 (17.5%) rewarmed spontaneously. These

* After attaining body temperature level of 26°C, further anesthesia is not required. Consequently the effects of anesthesia are minimal in these experimental results.

animals did not remian at 26 ± 1°C for the selected period of 18 to 20 hours and therefore cannot be considered as true survivors. When these 7 animals are excluded, and when the mortality during rewarming is not considered, then the actual mortality during prolonged hypothermia is 18 out of 33 (54.5%).

TABLE 1

Fate of 40* Animals Subjected to Prolonged Hypothermia

Survived:		17/40	42.5%
and rewarmed	10/40	25.0%	
rewarmed spontaneously	7/40	17.5%	
Died during prolonged hypothermia:		18/40	45.0%
in ventr. fibrillation	4/40	10.0%	
in asystole	14/40	35.0%	
Died during rewarming:		5/40	12.5%
in circulatory shock	3/40	7.5%	
in ventr. fibrillation	2/40	5.0%	

*This group excludes 3 animals that died of VF during the initial phase of cooling to 26 ± 1°C.

The accumulative mortality during prolonged hypothermia is shown in Table 2. Note that the mortality shows a continuous progression with time. Thus half way through the period approximately half of the animals that eventually died were dead. There is not an obvious critical time.

When the total number of 33 animals that were maintained at 26 ± 1°C is considered (excluding those that rewarmed spontaneously), approximately 50% were dead after 18 hours. Thus the time for 50% mortality, or the median lethal time (LT_{50}), during prolonged hypothermia at 26 ± 1°C may be taken to be in the vicinity of 18 hours.

TABLE 2

Cummulative Mortality of 33 Animals* which were Cooled and Maintained at 26 ± 1°C for a Period of 18 to 20 Hours.

Hours at 26 ± 1°C	Total No. Dead at the end of hr.	% of Total
2	1	3
4	2	6
6	5	15
8	6	18
10	8	24
12	11	33
14	11	33
16	12	36
18	16	49
20	18	55

*Excluding the 7 animals which rewarmed spontaneously

B. Heart Rate

Most animals exhibited a progressive decrease in heart rate while they were maintained under hypothermia even though the body temperature was kept fairly constant. This was especially striking in some of the animals that terminated in asystole. In these cases asystole occured after the heart rate was slowed to less than 30 beats/min. However this was not a universal finding and in some cases the results were complicated by the occurance of small (± 1°C) fluctuations in body temperature during the period of maintained hypothermia.

In general there was a tendency for a decrease in heart rate in all the animals maintained under hypothermia. This is clearly shown in the average values given in Table 3 for 14 animals which suriived 15 or more hours of prolonged hypothermia.

TABLE 3

Mean Heart Rates of 14 Animals Surviving 15 or More Hours of Prolonged Hypothermia at 26 ± 1°C

Hours at 26 ± 1°C	Mean Heart Rate	Hours at 26 ± 1°C	Mean Heart Rate
0	70.3	8	55.1
1	69.8	9	54.1
2	66.3	10	52.9
3	63.1	11	52.9
4	60.1	12	52.4
5	58.9	13	51.9
6	57.1	14	51.0
7	56.4	15	50.2

In all the animals for which data are available there was no evidence of any change in the location of the dominant pacemaker during cooling and maintaining under hypothermia. Thus the reported changes in heart rate reflect changes in the rate of the normal SA node pacemaker. Nevertheless in many animals there were occasional or even frequent ventricular extrasystoles while cooling and during the period of prolonged hypothermia.

It should be stressed that in the present experiments due to the long periods of observation required, all recordings were made at very slow paper speeds. This limits the interpretation of electrocardiographic changes.

C. Arterial Hematocrit

Measurements of arterial blood hematocrit during cooling and prolonged hypothermia were made in most animals. Data on the initial effect of cooling to 26 ± 1°C were collected in 35 animals and are given in Table 4. Complete data including a period of 18 to 20 hours under hypothermia were obtained from 14 animals and are given in Table 5. Hemoconcentration during the initial period of cooling was observed in 28 out of 35 animals. All 14 animals from which extensive data are available showed an

increase in hematocrit during the period they were maintained under prolonged hypothermia, and 9 of these exhibited an even further increase during rewarming. Unfortunately no extensive data are available on the exact time course of the increase in hematocrit during the period of maintained hypothermia.

TABLE 4

Changes in Blood Hematocrit with the Initial Period of Cooling to 26 ± 1°C (Data from 35 Animals)

Control 37 ± 1°C	Hypothermia 26 ± 1°C		Control 37 ± 1°C	Hypothermia 26 ± 1°C
40	45		47	52
34	33		63	70
38	46		43	46
31	34		35	51
42	47		53	68
38	52		58	53
41	57		45	60
44	54		30	50
45	55		44	51
48	48		37	44
38	55		48	57
36	36		39	60
39	40		53	60
42	55		60	70
30	46		45	70
56	56		40	57
56	51		40	54
57	57	Mean	43.9	52.6

TABLE 5

Changes in Blood Hematocrit with Initial Cooling, Maintained Hypothermia, and Rewarming (Data from 14 Animals)

Dog #	Control Before Cooling	Prolonged Hypothermia at 26 ± 1°C Start	Prolonged Hypothermia at 26 ± 1°C End	Control After Rewarming
3235	40	45	65	75
3244	38	46	52	55
3251	42	47	51	62
3258	38	52	59	63
3261	41	57	73	71
3017	42	55	63	64
3034	57	57	70	70
3037	47	52	52	55
3190	43	46	60	62
3194	35	51	56	56
3195	53	68	70	80
3196	58	53	48	60
3204	37	44	60	58
3213	40	57	70	70
Mean	43.6	52.1	60.6	64.4

The same trend for an increase in hematocrit with induction and maintenance of hypothermia was observed in all animals studied. There was no difference in this respect between the animals that died during prolonged hypothermia and those that survived. However the contribution of hemoconcentration to mortality cannot be fully evaluated due to the lack of data on hematocrits just prior to death.

The observed changes in hematocrit could not be interpreted with any degree of reliability without some information regarding the effect of prolonged pentobarbital anesthesia in the absence of hypothermia. Reports in the literature (6) indicate that dogs maintained under pentobarbital anesthesia for many hours show a

slight decrease in blood hematocrit. However, since this was a very important point in the interpretation of the results regarding the effect of hypothermia, a series of eight experiments were performed to study the effect of prolonged pentobarbital anesthesia alone on hematocrit. Results are given in Table 6.

TABLE 6

Blood Hematocrit During Prolonged Pentobarbital Anesthesia at Normothermic Temperatures (8 Animals)

	Time Hours				
Dog #	0	2	4	6	8
3271	44	47	47	49	49
3272	46	45	45	45	45
3273	47	47	51	49	49
3279	30	41	43	41	41
3280	49	46	46	46	45
3282	30	33	34	35	37
3285	38	47	46	48	48
3286	51	49	44	46	45
Mean	41.9	44.4	44.5	44.8	44.9

D. Arterial Blood pH

The well known development of acidosis during induction of hypothermia (7) was observed in all animals during the initial period of cooling. Values on 17 are given in Table 7. These pH values were obtained in spontaneously breathing animals.

TABLE 7

Arterial Blood pH During the Initial Period of Cooling (Data from 17 Animals)

Dog #	37 ± 1	32 ± 1	28 ± 1
3214	7.44	7.33	7.10
3235	7.34	7.13	7.24
3197	7.45	7.19	7.05
3037	7.37	7.23	7.17
3190	7.24	7.22	7.21
3194	7.36	7.22	7.11
3195	7.38	7.16	7.05
3196	7.36	7.26	7.19
3198	7.32	7.29	7.19
3200	7.37	7.21	7.14
3201	7.34	7.17	7.13
3203	7.37	7.17	7.17
3206	7.35	7.31	7.20
3209	7.44	7.15	7.12
3210	7.42	7.36	7.31
3211	7.37	7.16	7.13
3212	7.40	7.19	7.12
Mean	7.37	7.22	7.15

Positive pressure artificial respiration was given to all animals below 28°C. As discussed in previous Technical Reports (1,2), this is essential because during cooling under pentobarbital anesthesia respiration fails in a large proportion of animals beginning at body temperatures of 28 to 26°C.

Arterial blood pH at the beginning and again at the end of a period of 16 to 20 hours of maintained hypothermia at 26 ± 1°C was measured in 10 dogs and the data are given in Table 8. All the animals were respired artifically at a constant rate and

volume during the entire period. Artificial respiration was started for at least one hour before the control measurements were taken.

TABLE 8

Arterial Blood pH at the Beginning and End of a 15-20 Hour Period of Maintained Hypothermia at 26 ± 1°C (Data from 10 Animals)*

Dog #	Beginning	End
3244	7.49	7.28
3251	7.19	7.25
3258	7.12	7.14
3034	7.40	7.21
3037	7.49	7.42
3090	7.15	7.22
3194	7.45	7.45
3195	7.41	7.08
3204	7.45	7.10
3213	7.24	7.44
Mean	7.34	7.26

*All animals were kept under positive pressure artificial respiration with room air. Rate and depth of respiration were kept constant throughout the period.

Similar results were obtained in animals which survived over shorter periods of time. No significant correlation was found to exist between arterial pH or the degree of acidosis and survival under prolonged hypothermia.

E. Plasma Electrolytes

An attempt was made to measure changes in blood electrolytes during the experimental period. However the results were not consistent and were complicated by several technical difficulties. The most serious complication was the small but progressive hemolysis that occured during cooling and maintaining at hypothermic temperatures. Such systematic hemolysis introduced an important

element of unreliability in the results. In addition, the marked degree of hemoconcentration that occured in most animals suggested the possibility that there may be a water shift out of the intravascular compartment. This could account for the increase in the concentration of all values of plasma electrolytes observed in some animals.

On the basis of these preliminary observations it was decided to defer the study of the electrolyte pattern until measurements of plasma volume could be made at the same time.

DISCUSSION

A. Survival

The most significant objective of this study was to establish limits of tolerance under prolonged hypothermia at moderate temperature levels. Previous studies (1,2,3) indicate that only a very small proportion (less than 5%) of the animals terminate at temperatures above 26°C when hypothermia is induced acutely. The present results indicate that mortality increases progressively with time even though the body temperature is maintained at a fixed level. It is important to note (Table 2) that the increase in mortality is gradual and there is no critical time. In fact the data suggest strongly that if hypothermia were to be maintained for longer periods, all the animals would have eventually died.

An important difference was observed in the type of death that occured under prolonged hypothermia as compared with the acute procedure. Ventricular fibrillation is the dominant form of death during acute progressive hypothermia especially when death occurs at the higher temperatures (above 21°C). By contrast the majority of animals that died during prolonged hypothermia at 26 $\pm$ 1°C terminated in asystole (Table 1). This suggests that the factors responsible for death during acute exposure to cold and the initial period of cooling, are different from those which limit survival under prolonged hypothermia. Thus survival under prolonged hypothermia presents a new series of problems which require extensive investigation.

B. Heart Rate

A possible clue to the physiological alterations which precipitate death during prolonged hypothermia may be found in the observation that there is a progressive decrease in heart rate even though the body is maintained at a constant temperature level.

Cardiovascular reflex mechanisms are known to be relatively inactive at body temperatures of 26 ± 1°C. Therefore it seems reasonable to propose that the observed decrease in heart rate reflected a direct effect on the frequency of discharge of the SA node pacemaker fibers. Such a decrease in the pacemaker rate could result from an increased intracellular accumulation of sodium ions. This interpretation is consistent with the expectation that the metabolically dependent mechanisms responsible for sodium efflux from the cells would be inhibited by low temperatures. Thus the intracellular accumulation of sodium may increase progressively even though the heart temperature is maintained at a constant hypothermic level. This in turn would result in a progressive slowing in heart rate. Data on tissue electrolyte levels are required to substantiate this hypothesis.

In this connection it would be of interest to test whether the use of artificial electronic pacemakers would prolong survival under maintained hypothermia.

C. Hematocrit

The increased hemoconcentration is the most striking and most consistent change observed during cooling and prolonged hypothermia. It is clear from Table 5 that the increase in hematocrit is progressive and it is not reversible by rewarming.

Data on anesthetized normothermic dogs maintained for several hours (Table 6) indicate that the increase in hematocrit observed during hypothermia is not due to anesthesia or time alone. These results on normothermic dogs are in essential agreement with published reports on the effect of prolonged pentobarbital anesthesia alone on hematocrit (6).

The factors responsible for the observed increase in hematocrit are at present unknown. Hemoconcentration may result from: a) increase in the RBC volume released into the general circulation from RBC storage sites (e.g. the spleen) b) decrease in the total plasma volume due to a water shift out of the intravascular compartment or c) decrease in the circulating plasma volume due to "trapping" of plasma in small peripheral vessels or in general due to a change in the relative hematocrit between small and large blood vessels.

There is some published evidence of plasma "trapping" in small blood vessels during acute hypothermia (8). The effect of prolonged hypothermia may be a further exaggeration of this change. However other factors should be excluded.

Regardless of its cause, the increased hemoconcentration and the resulting increase in blood viscosity must affect greatly the circulatory dynamics under prolonged hypothermia. Tissue perfusion is undoubtedly reduced under these conditions.

It would be of particular interest to determine whether induced hemodilution by infusions of saline or plasma could affect survival under prolonged hypothermia. However animals with spontaneously low initial hematocrits did not appear to tolerate prolonged hypothermia any better than animals with spontaneously high hematocrits.

D. Arterial pH

The observed acidosis during the initial period of cooling is well known and deserves no further discussion.

It is noteworthy that under constant ventilation, there are no consistent changes in arterial pH during maintained hypothermia (Table 8). In general there was no significant correlation between arterial blood pH, or the degree of hypothermic acidosis, with survival under prolonged hypothermia. Thus under the conditions of the experiments reported here, the acid-base balance does not appear to play a significant role in determining survival.

E. Conclusions

The following conclusions can be drawn from the experimental results presented.

1. During prolonged hypothermia at moderate temperatures (26 $\pm$ 1°C) there is a progressive mortality with time. No critical time period appears to exist.

2. Survival during prolonged hypothermia appears to depend upon factors different from those which determine survival during the acute induction of the hypothermic state.

3. The SA node pacemaker rate decreases gradually during prolonged hypothermia even though the heart temperature is maintained at a nearly constant level. However there is no definite association between the degree of bradycardia and survival time under maintained hypothermia.

4. There is a striking increase in hematocrit during cooling which is maintained under hypothermia. This is not due to anesthesia or time and it is not reversible with rewarming. The factors responsible for the hemoconcentration are unknown.

5. The degree of hypothermic acidosis is not consistently exaggerated by maintained hypothermia and does not appear to play a role in survival during prolonged hypothermia.

6. Plasma electrolyte changes during prolonged hypothermia are inconsistent and cannot be interpreted without concurrent information on total plasma volume.

7. Additional studies are needed to further elucidate the factors limiting survival under prolonged hypothermia and to evaluate their relative contribution. In particular studies are needed on: a) effect of artificial pacemakers, b) evaluation of myocardial contractility, c) measurements of changes in plasma volume, and d) concurrent data on plasma electrolyte values.

REFERENCES

1. Hegnauer, A.H. Ventricular Fibrillation in the Hypothermic Dog. WADC Technical Report 55-79, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio, February 1955.

2. Hegnauer, A.H. and E.T. Angelakos. Pharmacological Control of Ventricular Fibrillation. WADC Technical Report 59-125. Aero Medical Laboratory, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio, May 1959.

3. Angelakos, E.T. "Distribution of Terminal Temperatures in Hypothermic Dogs" Proc. Soc. Exp. Biol. & Med Vol. 97, p. 107, 1958.

4. Cotlove, E., H.V. Trantham and R.L. Bowman. "An Instrument and Method for Automatic, Rapid, Accurate and Sensitive Titration of Chloride in Biological Samples". J. Lab. & Clin. Med. Vol. 51, p. 461, 1958.

5. Bachra, B.N., A. Dauer and A.E. Sobel. "The Complexometric Titration of Micro and Ultramicro Quantities of Calcium in Blood, Serum, Urine and Inorganic Salt Solutions". Clin. Chem. Vol. 4, p. 107, 1958.

6. Nash, C.B., F. Davis and R.A. Woodbury. "Cardiovascular Effects of Anesthetic Doses of Pentobarbital Sodium". Am. J. Physiol. Vol. 185, p. 107, 1956.

7. Covino, B.G. and A.H. Hegnauer. "Electrolytes and pH Changes in Relation to Hypothermic Ventricular Fibrillation" Circulation Research, Vol. 3, p. 575, 1955.

8. D'Amato, H.E. and A.H. Hegnauer. "Blood Volume in the Hypothermic Dog". Am. J. Physiol., Vol. 173, p. 100, 1953

Aerospace Medical Division, 6570th Aerospace Medical Research Laboratories, Wright-Patterson AFB, Ohio. Rpt. No. AMRL-TDR-62-130. STUDIES AT MODERATE HYPOTHERMIC TEMPERATURES OF FACTORS AFFECTING SURVIVAL UNDER PROLONGED HYPOTHERMIA. Final report, Nov 62, v + 15 pp. incl. tables, and 8 refs. Unclassified report

The purpose of this investigation was to study the mortality of anesthetized dogs maintained under hypothermia for many hours, and to evaluate the factors responsible for death under these conditions. Data are presented on technically successful experiments in 40 dogs maintained at 26 ±1°C. Among the animals that died during maintained hypothermia, mortality was progressive with no apparent critical time. The median lethal time at 26 ±1°C is estimated to be 18 hours. There was a progressive decrease in the average normal pacemaker heart rate in animals maintained at a relatively fixed hypothermic temperature. Values of arterial hematocrit showed a progressive and striking increase during cooling, maintained under hypothermia, and rewarming. Neither degree of hypothermic acidosis nor plasma electrolyte changes were altered consistently during maintained hypothermia. The factors limiting survival under prolonged hypothermia are different from those involved in the acute induction of the hypothermic state.

(over)

UNCLASSIFIED

1. Hypothermic
2. Exposure
3. Heart
4. Laboratory animals
5. Biothermal Studies
- I. AFSC Project 7222, Task 722204
- II. Biomedical Laboratory
- III. Contract AF 33(616)-6767
- IV. Boston University School of Medicine, Boston, Mass.
- V. Angelakos, E. T., M. D.
- VI. In ASTIA collection
- VII. Aval fr OTS: $0.75

UNCLASSIFIED

Aerospace Medical Division, 6570th Aerospace Medical Research Laboratories, Wright-Patterson AFB, Ohio. Rpt. No. AMRL-TDR-62-130. STUDIES AT MODERATE HYPOTHERMIC TEMPERATURES OF FACTORS AFFECTING SURVIVAL UNDER PROLONGED HYPOTHERMIA. Final report, Nov 62, v + 15 pp. incl. tables, and 8 refs. Unclassified report

The purpose of this investigation was to study the mortality of anesthetized dogs maintained under hypothermia for many hours, and to evaluate the factors responsible for death under these conditions. Data are presented on technically successful experiments in 40 dogs maintained at 26 ±1°C. Among the animals that died during maintained hypothermia, mortality was progressive with no apparent critical time. The median lethal time at 26 ±1°C is estimated to be 18 hours. There was a progressive decrease in the average normal pacemaker heart rate in animals maintained at a relatively fixed hypothermic temperature. Values of arterial hematocrit showed a progressive and striking increase during cooling, maintained under hypothermia, and rewarming. Neither degree of hypothermic acidosis nor plasma electrolyte changes were altered consistently during maintained hypothermia. The factors limiting survival under prolonged hypothermia are different from those involved in the acute induction of the hypothermic state.

(over)

UNCLASSIFIED

1. Hypothermic
2. Exposure
3. Heart
4. Laboratory animals
5. Biothermal Studies
- I. AFSC Project 7222, Task 722204
- II. Biomedical Laboratory
- III. Contract AF 33(616)-6767
- IV. Boston University School of Medicine, Boston, Mass.
- V. Angelakos, E. T., M. D.
- VI. In ASTIA collection
- VII. Aval fr OTS: $0.75

UNCLASSIFIED